Type II endoleak is an enigmatic and unpredictable marker of worse outcome after endovascular aneurysm repair

Enrico Cieri, MD, PhD,[a] Paola De Rango, MD, PhD,[a] Giacomo Isernia, MD,[a] Gioele Simonte, MD,[a] Andrea Ciucci, MD,[a] Gianbattista Parlani, MD,[a] Fabio Verzini, MD, PhD, FEBVS,[a] and **Piergiorgio Cao, MD, FRCS,[b]** *Perugia and Rome, Italy*

Background: **This study analyzed predictors and the long-term consequence of type II endoleak in a large series of elective endovascular abdominal aneurysm repairs (EVARs).**

Methods: **Baseline characteristics and operative and follow-up data of consecutive patients undergoing EVAR were prospectively collected. Patients who developed type II endoleak according to computed tomography angiography and those without type II endoleak were compared for baseline characteristics, mortality, reintervention, conversion, and aneurysm growth after repair.**

Results: **In 1997-2011, 1412 consecutive patients (91.4% males; mean age, 72.9 years) underwent elective EVAR and were subsequently followed up for a median of 45 months (interquartile range, 21-79 months). Type II endoleak developed in 218. Adjusted analysis failed to identify significant independent predictors for type II endoleak with the exception of age (odds ratio, 1.03; 95% confidence interval, 1.01-1.05; $P = .003$) and intraluminal thrombus (odds ratio, 0.69; 95% confidence interval, 0.53-0.92; $P = .010$). Type II endoleak rates were comparable regardless of the device model. Late aneurysm-related survival was comparable (98.4% vs 99.5% at 60 months; $P = .73$) in patients with and without type II endoleak. However, at 60 months after EVAR, rates of aneurysm sac growth >5 mm (35.3% vs 3.3%; $P < .0001$) were higher in patients with type II endoleak. Cox regression identified type II endoleak as an independent predictor of aneurysm growth along with age and cardiac disease. The presence of type II endoleak led to reinterventions in 40% of patients and conversion to open surgery in 8%. However, assessment of these patients after reintervention showed similar 60-month freedom rates of persisting type II endoleak (present in more than two after computed tomography angiography scan studies) among those with and without reinterventions (49.8% vs 45.6%; $P = .639$). Aneurysm growth >5 mm persisted with comparable rates in type II endoleak patients after reintervention and in those who remained untreated (42.9% vs 57.4% at 60 months; $P = .117$).**

Conclusions: **Reintervention for type II endoleak was common in our practice, yet such intervention did not reliably prevent the continued expansion of the abdominal aortic aneurysm. Our data indicate type II endoleak appears to be a marker of EVAR failure that is difficult to predict and treat effectively. (J Vasc Surg 2014;59:930-7.)**

The clinical relevance and natural history of type II endoleaks, the most common category of endoleaks recorded after an endovascular abdominal aneurysm repair (EVAR), remain largely unsettled. Currently, there is no evidence to support a single optimal threshold for intervention or the best treatment strategy in patients with isolated type II endoleak.[1] The long-term success for treating type II endoleak is not ideal, because studies show endoleak persistence or continuing abdominal aortic aneurysm (AAA) growth after secondary interventions for type II endoleak. However, data are limited on whether type II endoleaks are predictable, preventable, and treatable, and consistent debate remains.[2-4]

The objective of this study was to analyze promoting factors, associated outcomes, and effect of reintervention in type II endoleak in EVAR patients.

METHODS

The study analyzed consecutive patients who underwent elective EVAR for infrarenal AAAs between April 1997 and December 2011 and were entered in a prospective database. Collected data included preoperative demographics, comorbidities, morphology, intraoperative details, and follow-up information. Patients treated with fenestrated stent grafts or treated emergently for AAA rupture were excluded.

EVAR was performed by a dedicated team, with the patient under general or local anesthesia. Different device models were used depending on the aortoiliac morphology, stent graft availability, and operator preferences. Devices

From the Unit of Vascular and Endovascular Surgery, Hospital S. Maria della Misericordia, University of Perugia, Perugia[a]; and the Unit of Vascular and Endovascular Surgery, Department of Cardiosciences, Hospital S. Camillo-Forlanini, Rome.[b]

Author conflict of interest: none.

Presented as a rapid session oral presentation at the 2013 Vascular Annual Meeting of the Society for Vascular Surgery, San Francisco, Calif, May 30-June 1, 2013.

Reprint requests: Paola De Rango, MD, PhD, Vascular and Endovascular Unit, Hospital S. Maria Misericordia, Loc. S. Andrea delle Fratte, 06134 Perugia, Italy (e-mail: plderango@gmail.com).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214/$36.00

http://dx.doi.org/10.1016/j.jvs.2013.10.092

were defined as "new generation" if introduced after 2004 and currently in use.[5]

After EVAR, patients were scheduled for serial follow-up imaging with duplex ultrasound and computed tomography angiography (CTA) scan at 1 month, 6 months, and yearly thereafter. Postoperative surveillance was performed using multiphasic CTA with 3-mm cuts, including imaging before the administration of contrast material, after the intravenous administration of contrast material in the arterial phase (arterial phase), and in postcontrast delayed phase (venous phase). The TeraRecon Aquarius dedicated digital workstation (Terarecon, Foster City, Calif) was used for CTA scan imaging analysis and three-dimensional reconstructions.

Preoperative and postoperative CTA scans were compared, and aneurysm sac diameter and endoleak was recorded. Maximum aneurysm diameter was determined by the shortest diagonal of the cross-sectional CTA image perpendicular to the centerline of flow on CTA reconstructions. Diameter changes >5 mm between two studies after CTA were considered significant for sac growth based on the Standardized Reporting Practices in Vascular Surgery.[6]

Type II endoleak was defined as blood flow outside the stent graft but within the aneurysm sac caused by retrograde refilling from aortic side branches as assessed on imaging.[6] Diagnosis of type II endoleak was made using contrast imaging with delayed-enhancement phase. Duplex ultrasound findings were always retested with a contrast-enhanced CTA scan or angiography before treatment (conservative, strict surveillance, embolization) was planned. Arterial and delayed-phase imaging was used in the post-EVAR CTA protocol for type II endoleak detection. Reintervention for type II endoleak was performed at the discretion of the attending surgeon, but treatment was applied when AAAs showed a persisting type II endoleak (present in two or more post-EVAR CTA scans) associated with diameter growth >5 mm after EVAR.

Type of treatment was individualized to aneurysm anatomy and endoleak source. Lumbar endoleaks were generally accessed through retrograde cannulation of iliolumbar arteries from hypogastric arteries, followed by spirals and coils embolization.[7] Direct sac puncture with a spinal needle using CTA guidance and coil embolization was an alternative treatment performed less frequently.

Type II inferior mesenteric artery (IMA) endoleaks were commonly embolized by retrograde access to the IMA through the arc of Riolan or the marginal artery after the middle colic artery was accessed through the superior mesenteric artery.[3,8]

Primary outcome measures were mortality and freedom from aneurysm growth. Secondary outcome measures included aneurysm-related mortality, endoleak persistence, and the rate of reintervention and conversion to open surgery.

Statistical analysis. Patients were separately analyzed according to the presence or absence of type II endoleak. Patients with type II endoleak were further categorized according to treatment applied, and those receiving or not receiving reintervention after EVAR were compared for long-term outcomes.

Categoric factors are summarized using frequencies and percentages, and continuous measures are reported using means and standard deviations or the median and interquartile range (IQR), when appropriate. Comparisons between groups were analyzed for significance using χ^2 tests, analysis of variance, *t*-test, or the Fisher exact test, when appropriate.

The independent associations with type II endoleak and aneurysm growth were tested with Cox regression analyses. The most reported and clinically relevant morphology and patient-related predictors of aneurysm growth and endoleak in literature were introduced into the Cox model. Backward stepwise selection was used to exclude confounders and select significant predictors retained in the final model. Results are expressed as odds ratios (ORs) and correspondent 95% confidence intervals (CIs). To estimate survival, aneurysm-related survival rates of reintervention, conversion, and aneurysm growth, Kaplan-Meier analysis with log-rank test to determine significance of comparison was used. Significance was assumed at $P < .05$. Analyses were performed using SPSS 20.0 software (IBM Corp, Armonk, NY).

RESULTS

During the study period, 1412 (91.4% males; mean age, 72.9 years) consecutive elective EVARs were performed with different device models. At a median follow-up of 45 months (IQR, 21-79 months), 218 type II endoleaks were recorded. Baseline characteristics of patients with and without type II endoleak are reported in Table I.

Predictors of type II endoleak. Type II endoleak rates were comparable regardless of the type of device, but there was a tendency for lower rates with the most recent generation devices (Table II). Cox regression analysis adjusted for covariates (age, sex, diabetes, smoking, hypertension, hyperlipidemia, coronary disease, chronic obstructive pulmonary disease, peripheral artery disease, AAA diameter, use of anticoagulants, intraluminal thrombus) identified old age (continuous variable) as a positive predictor of type II endoleak (OR, 1.03; 95% CI, 1.01-1.05; $P = .003$). The presence of intraluminal aortic thrombus was the only indicator of decreased type II endoleak occurrence (OR, 0.69; 95% CI, 0.53-0.92; $P = .010$).

Survival and aneurysm-related survival. All-cause survival was higher in patients who developed type II endoleak: 88.4% vs 79.7% at 36 months and 76.1% vs 67% at 60 months in patients with and without type II endoleak, respectively ($P = .01$).

Aneurysm-related survival was indeed comparable: rates were 100% vs 99.5% at 36 months and 98.4% vs 99.5% at 60 months for patients with and without type II endoleak, respectively ($P = .730$).

Overall, 17 aneurysm ruptures occurred over a median of 55 months (IQR, 42-75 months) after EVAR and due to development of type I (n = 5), II (n = 4), and

Table I. Baseline characteristics and demographics of patients with and without type II endoleak

Variable[a]	*Type II endoleak (n = 218)*	*No type II endoleak (n = 1194)*	P
Age, years	73.7 ± 6.9	72.8 ± 7.8	.106
Males	194 (89.0)	1096 (91.8)	.113
Hypertension	171 (78.4)	913 (76.5)	.295
CAD	102 (46.8)	555 (46.5)	.496
Hyperlipidemia	69 (31.7)	428 (35.8)	.132
PAD	21 (9.6)	162 (13.6)	.066
Diabetes	28 (12.8)	147 (12.3)	.45
COPD	101 (46.3)	595 (49.8)	.190
Chronic renal failure	28 (12.8)	173 (14.5)	.301
Smoking	113 (51.8)	704 (59.0)	.03
Cerebrovascular disease	25 (11.5)	168 (14.1)	.179
Anticoagulants	19 (8.7)	82 (6.9)	.201
Antiplatelet	118 (54.1)	594 (49.7)	.132
Aneurysm diameter, mm	53 (49-58)	53 (50-59)	.256
Neck length, mm	25 (20-30)	23 (18-30)	.072
Neck diameter, mm	23 (21-24)	23 (22-25)	.155
Neck thrombus[b]	5 (2.3)	83 (7.0)	.004
Aortic thrombus[c]	101 (46.3)	594 (49.7)	.196

CAD, Coronary artery disease; *COPD,* chronic obstructive pulmonary disease; *IQR,* interquartile range; *PAD,* peripheral artery disease.
[a]Continuous data are shown as the mean ± standard deviation or median (IQR) and categoric data are shown as number (%).
[b]A continuous parietal layer of thrombus at least three-fourths the circumference in one section.
[c]Severe if >50% of the circumference.

Table II. Type II endoleak distribution by device models

Device	*No.*	*Type II endoleak (n = 218), No. (%)*	*No type II endoleak (n = 1194), No. (%)*	P
Zenith[a]	610	95 (43.6)	515 (43.1)	.480
Excluder[b]	233	38 (17.4)	195 (16.3)	.376
Endurant[c]	73	4 (1.8)	69 (5.8)	.007
Talent[c]	167	21 (9.6)	147 (12.2)	.164
AneuRx[c]	235	39 (17.9)	196 (16.4)	.326
Anaconda[d]	53	11 (5.0)	42 (3.5)	.182
Fortron[e]	35	10 (4.6)	25 (2.1)	.033
Others	6			
New devices	882	129 (59.2)	753 (63.1)	.155

[a]Cook Medical, Bloomington, Ind.
[b]W. L. Gore & Associates, Flagstaff, Ariz.
[c]Medtronic, Santa Rosa, Calif.
[d]Terumo Vascutek, Inchinnan Renfrewshire, Scotland, UK.
[e]Johnson & Johnson-Cordis Corporation, Bridgewater, NJ.

III (n = 2) endoleak, migration (n = 4), stent fracture (n = 2), or iliac aneurysm rupture. The four ruptures in the patients with type II endoleak occurred at 42, 43, 55, and 74 months after EVAR. In one patient, rupture occurred 1 year after unsuccessful IMA embolization and in another, type I endoleak was also recorded at the time of rupture.

Aneurysm growth at follow-up. Rates of freedom from aneurysm growth are shown in Fig 1. There was a significant higher proportion of growth >5 mm in the group of aneurysms with type II endoleak than in those without: 15.5% vs 1.9% at 36 months and 35.3% vs 3.3% at 60 months ($P < .0001$).

Cox regression analysis (adjusting for age, coronary disease, diabetes, smoking, hypertension, chronic renal failure, peripheral artery disease, type II endoleak, hyperlipidemia, AAA diameter, on anticoagulant) confirmed a strong independent association between type II endoleak and the likelihood of aneurysm growth after EVAR (OR, 4.93; 95% CI, 3.62-6.72; $P < .0001$). Less relevant independent association with aneurysm growth was also shown for old age (OR, 1.03; 95% CI, 1.01-1.06; $P = .006$) and history of coronary disease (OR, 1.49; 95% CI, 1.09-2.03; $P = .012$).

Reintervention during follow-up. During follow-up, 52 patients with type II endoleak associated with aneurysm growth underwent reinterventions, and in 15, more than one reintervention was undertaken. Details of reinterventions are reported in Tables III and IV.

Reintervention rates were more common in patients with type II endoleak compared with the others. Freedom from reintervention rates were 79.0% vs 97.5% at 36 months and 60.2% vs 94.9% at 60 months for patients with and without type II endoleak, respectively ($P < .0001$; Fig 2).

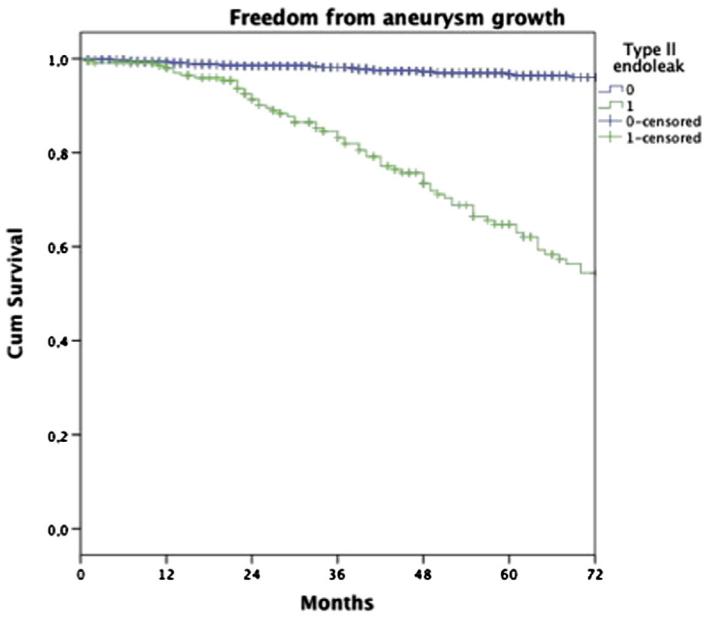
A total of 31 late conversions to open surgery were performed, 21 of which were in the type II endoleak group. In 11 patients with type II endoleak, a previous reintervention had already been performed with unsuccessful outcome before conversion: 1 IMA embolization, 4 lumbar embolizations, and 6 additional extension cuffs. Conversion rates were significantly higher in patients who displayed type II endoleak: freedom rates were 97% vs 99.3% at 36 months and 92.2% vs 98.9% at 60 months for patients with and without type II endoleak ($P < .0001$).

Twenty-two aneurysms with type II endoleak of the 52 that received reintervention continued to enlarge. Aneurysm growth rates after reintervention were comparable between patients with type II endoleak treated with reintervention and those without treatment: 28.6% vs 39.5% at 36 months, 42.9% vs 57.4% at 60 months ($P = .117$; Fig 3).

Kaplan-Meier estimates of freedom from type II endoleak persistence after reintervention also showed proportions comparable to those of patients with type II endoleak without reintervention: 65.7% vs 64% at 36 months and 49.8% vs 45.6% at 60 months in patients with type II endoleak receiving reintervention and those left untreated, respectively ($P = .639$). The evolution of type II endoleak is summarized in Fig 4.

DISCUSSION

Despite the high prevalence (10% to 30% in most series) and the increasing number of studies investigating the nature and evolution, type II endoleaks remain obscure events that can be assumed as generic markers of negative prognosis after EVAR.[3,9-12] Precise characterization of type II endoleaks as "benign" or "threatening" events is unreliable, because it is likely that multiple adverse factors, with different clinical relevance, are all similarly labeled as



	Months	6	12	24	36	48	60
No endoleak	n° at risk	984	927	763	615	452	332
	Survival %	99.7	99.3	98.4	98.1	97.2	96.7
Type II endoleak	n° at risk	207	188	160	130	105	78
	Survival %	99.1	98.0	92.5	84.5	75.7	64.7

Fig 1. Freedom from aneurysm growth >5 mm in patients with and without type II endoleak according to Kaplan-Meier analysis. Curves are displayed up to a value of a standard error of <10%.

Table III. Reintervention for type II endoleak treatment (35 patients undergoing single reintervention)

Type of reintervention	*Patients (n = 35), No. (%)*
IMA embolization	10 (29)
Lumbar embolization	7 (20)
Aneurysm sac embolization	1 (3)
CT-guided sac embolization	7 (20)
Hypogastric	
Embolization + iliac cuff	8 (23)
Surgical ligature	1 (3)
IMA laparoscopic clipping	1 (3)

CT, Computed tomography; *IMA,* inferior mesenteric artery.

"type II endoleaks" because retrograde flow refilling of the aortic sac is their more immediate manifestation. However, given the large variability, any unique characterization of type II endoleak allows for fallacious optimal treatment. The multiple implications of the commonly labeled type II endoleak are also shown by variability in reporting in published studies: "early," "delayed," "persisting," "spontaneously disappearing," "pressurized," but also "intermittent" or "recurrent" type II endoleak have been identified, without clear indication because the variable behavior is indeed related to missing information.[13]

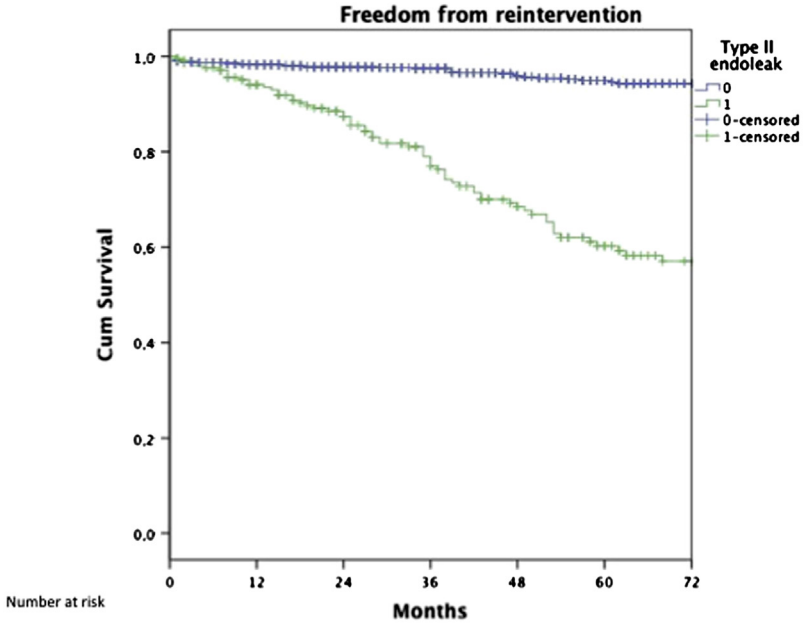
This study failed to show relevant modifiable predictors of type II endoleak. The only independent variables selected with Cox analysis were advanced age and intraluminal thrombus. A number of other studies also suggested aortic thrombus load and patency of aortic branches as the most common important anatomic predictors of type II endoleaks.[12,14-16] However, even if these suggested predictors could be identified, there is little chance to adjust their effect on risk: these are all nonmodifiable factors that do not allow effective strategies to preclude or effectively reverse type II endoleaks, which therefore appear to be unpreventable occurrences. Studies also showed that preventive coil/embolization of patent aortic branches before EVAR may be of little benefit on the occurrence of type II endoleak and aneurysm shrinkage.[16] Furthermore, the categorization of aortic thrombus lacks standardization, whereas the exact number of patent aortic branches is not always recognized in routine preoperative CTA imaging.

Even if potential causes of type II endoleak are treated, there is little evidence that treatment is effective. In this series, 35.3% of patients with type II endoleak had a >5 mm growing sac at 60 months. Of these, only a minority was successfully treated, because 22 of 52 continued to grow after reintervention. Furthermore, almost half of the treated type II endoleaks persisted after treatment. The quite high number of late conversion to open

Table IV. Reinterventions for type II endoleak treatment (15 patients undergoing more than one reintervention)

Patient	*First reintervention*	*Secondary reintervention*	*Tertiary reintervention*
1	Lumbar embolization	IMA embolization	
2	Lumbar embolization	IMA embolization	Hypogastric embolization + iliac cuff
3	Hypogastric embolization + iliac cuff	CT-guided embolization	…
4	CT-guided embolization	IMA embolization	…
5	IMA embolization	Sac embolization	…
6	Angiography (embolization attempt)	Surgical sac suture around stent graft	…
7	Angiography (embolization attempt)	CT-guided embolization	…
8	Angiography (embolization attempt)	CT-guided embolization	…
9	Angiography (embolization attempt)	CT-guided embolization	…
10	Angiography (embolization attempt)	CT-guided embolization	…
11	Angiography (embolization attempt)	Hypogastric embolization + iliac cuff	…
12	Angiography (embolization attempt)	Hypogastric embolization + iliac cuff	…
13	Angiography (embolization attempt)	Transcaval embolization	…
14	Angiography (embolization attempt)	Transcaval embolization	Hypogastric embolization + iliac cuff
15	Angiography (embolization attempt)	Lumbar embolization	…

CT, Computed tomography; *IMA,* inferior mesenteric artery.

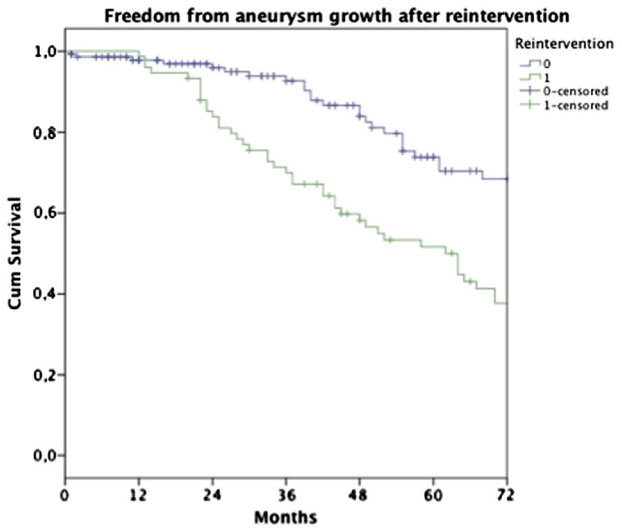


	Months	6	12	24	36	48	60
No endoleak	n° at risk	983	882	785	606	456	355
	Survival %	98.7	98.3	97.8	97.5	96.3	94.9
Type II endoleak	n° at risk	200	181	152	116	89	66
	Survival %	97.6	94.0	88.5	79.0	68.5	60.2

Fig 2. Freedom from reintervention in patients with and without type II endoleak according to Kaplan-Meier analysis. Curves are displayed up to a value of a standard error of <10%.

surgery, approaching 8% at 5 years, additionally reflected the higher risk of unfavorable outcome exposure in type II endoleak aneurysms (5-year conversion rate of 2% in those without type II endoleak). Therefore, only a few patients with type II endoleak would benefit from treatment.

Other experienced centers reported similar unsuccessful treatment outcome for type II endoleak. Aziz et al[2] analyzed results at 23 months after type II endoleak embolization and found no differences in the rates of aneurysm growth before and after type II endoleak treatment. Follow-up imaging of recurrent or persisting endoleak were recorded in 72% of patients.[2] Sarac et al[3] demonstrated that at 5 years, 56.3% of patients who underwent embolization for type II endoleak continued to experience



	Months	6	12	24	36	48	60
No reintervention	n° at risk	136	121	111	88	71	49
	Survival %	98.6	97.8	96.9	93.8	86.6	73.9
Reintervention	n° at risk	75	74	62	51	40	31
	Survival %	100	98.7	85.2	71.4	59.8	51.7

Fig 3. Freedom from continuing aneurysm growth >5 mm in patients with type II endoleak undergoing reintervention and remaining untreated according to Kaplan-Meier analysis. Curves are displayed up to a value of a standard error of <10%.

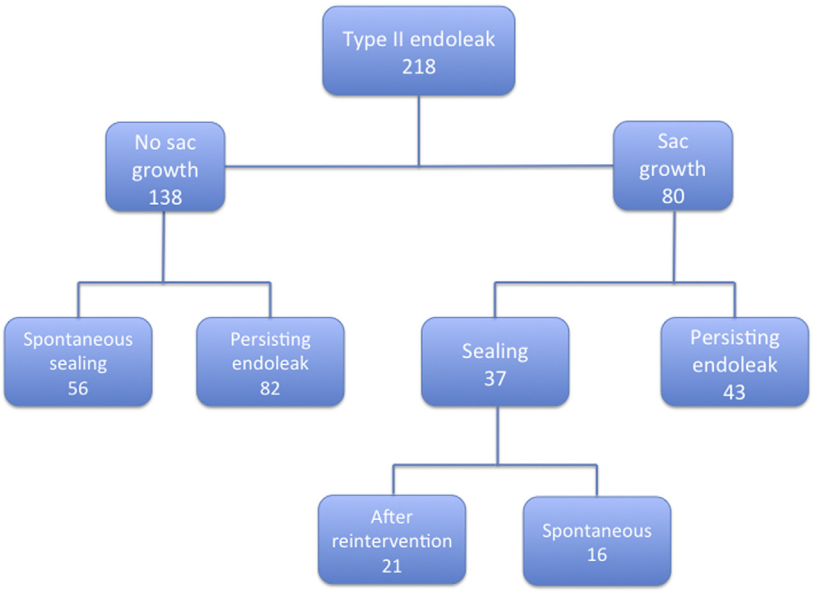


Fig 4. Summary of interventions for type II endoleaks.

sac growth, and a significant number of patients required more than one embolization procedure, regardless of material and techniques used for treatment. Gallagher et al[15] found that 56% of patients with type II endoleak required multiple reinterventions in the attempt to prevent aneurysm enlargement; however, embolization (specifically lumbar more than IMA embolization) carried a low midterm success rate.

It may be hypothesized that most reinterventions for type II endoleak are unsuccessful because they are wrongly applied. The source of refilling might not be properly identified, and incongruous treatment is often performed.

Type II endoleaks might be misdiagnosed, with more serious type (I or III) endoleaks not properly identified on imaging. The correlation frequently shown between persisting type II endoleak and subsequent type I or III endoleak development may not be the consequence of lack of sealing due to aneurysm enlargement prompted by type II endoleak but might be due to type I refilling that was originally missed but became evident on late imaging. One of the four ruptures in aneurysms with type II endoleak in our series was associated with evidence of type I endoleak at the time of rupture. Whether this type II endoleak was an originally missed type I or III endoleak remains questionable but unclear to us on performed imaging. Today, CTA with three-dimensional reconstruction is the best technique to use to monitor EVAR patients. However, imaging needs to be improved to better define type II endoleaks.

Although most of our type II reinterventions had unsuccessful morphology outcomes (persistence of endoleak or sac growth), type II endoleak was not associated with increased aneurysm mortality or rupture risk, as also supported by most other studies.[1,3,11,15] Whether this was a consequence of an effective aggressive reintervention strategy or there was a tendency toward overtreatment for most benign type II endoleaks continues to be debated.

An unexpected finding of this study was the better all-cause long-term survival in patients with type II endoleak, although they were ~1 year older than the others. We do not have a clear explanation for this apparent counterintuitive difference that might be due to chance or related to the presence of other unrecognized comorbidities or diseases (eg, cancer) with a higher prothrombotic burden that could have influenced the sealing of the aortic sac and the higher mortality in patients without type II endoleak.

Literature is conflicting in reporting the clinical relevance and thereby the decision for the treatment of type II endoleak,[2,3,9,15,17-22] with most recent studies[2,3,9,15] questioning the benignity previously suggested.[17] Gelfand et al[17] in 2006 published a meta-analysis of 10 EVAR trials involving 2617 patients and found no association between aneurysm rupture and type II endoleak. They suggested that type II endoleaks be followed up expecting that most (at least one-half) will disappear spontaneously. Definitive elective treatment was recommended only if type II endoleak persisted for >12 months or pulsatile sac or sac enlargement of >5 mm was detected during a 6-month period.[17] More recently, however, Sarac et al[3] analyzed the long-term outcome of type II endoleak and advocated early treatment for any type II endoleak associated with an increase in sac size.

The lack of consensus on how to currently approach this type of endoleak was shown by a more recent meta-analysis of EVAR studies performed by Karthikesalingam et al.[1] Articles were classified by the threshold for intervention on isolated type II endoleak as conservative, selective (intervention for >5 mm sac expansion or persistent for >6 months), or aggressive (any type II endoleak or persistent for >3 months). Of the overall 231 endoleaks recorded, 56 were treated at an aggressive threshold, 104 at a selective threshold, and 71 at a conservative threshold. Meta-regression demonstrated that no evidence for any strategy, compared with a conservative approach, reduced sac expansion or improved sac shrinkage.[1]

The inconsistent current management of type II endoleak reflects large uncertainty because of the multiple implications of type II endoleaks. Type II endoleak may represent only a marker of some other unfavorable condition after EVAR; otherwise, total sac exclusion should have been obtained. Currently, type II endoleak cannot be straightforwardly defined as predictable, preventable, or that its treatment alters outcomes.

CONCLUSIONS

Occurrence and consequences of type II endoleak are challenging to manage, and treatment with reinterventions often results in failure. The type II endoleak remains enigmatic and likely represents a common single marker of various unidentified complications of EVAR sharing multiple underlying implications. The correct origin of type II endoleak refilling cannot always be identified and therefore effectively treated. Current knowledge is lacking the true meaning of type II endoleak, and further effort should be encouraged to define this apparently benign complication of EVAR.

We thank Francesca Zannetti for editing and language support.

AUTHOR CONTRIBUTIONS

Conception and design: EC, PDR
Analysis and interpretation: EC, PDR, GS, GI, PC
Data collection: GS, GI, AC, FV, GP
Writing the article: EC, PDR, GS, GI
Critical revision of the article: EC, PDR, AC, FV, GP, PC
Final approval of the article: EC, PDR, GS, GI, AC, GP, FV, PC
Statistical analysis: EC, PDR, GS, GI
Obtained funding: Not applicable
Overall responsibility: EC
PDR and EC participated equally and share first authorship.

REFERENCES

1. Karthikesalingam A, Thrumurthy SG, Jackson D, Choke E, Sayers RD, Loftus IM, et al. Current evidence is insufficient to define an optimal threshold for intervention in isolated type II endoleak after endovascular aneurysm repair. J Endovasc Ther 2012;19:200-8.
2. Aziz A, Menias CO, Sanchez LA, Picus D, Saad N, Rubin BG, et al. Outcomes of percutaneous endovascular intervention for type II endoleak with aneurysm expansion. J Vasc Surg 2012;55:1263-7.
3. Sarac TP, Gibbons C, Vargas L, Liu J, Srivastava S, Bena J, et al. Long-term follow-up of type II endoleak embolization reveals the need for close surveillance. J Vasc Surg 2012;55:33-40.
4. Parry DJ, Kessel DO, Robertson I, Denton L, Patel JV, Berridge DC, et al. Type II endoleaks: predictable, preventable, and sometimes treatable? J Vasc Surg 2002;36:105-10.

5. Cieri E, De Rango P, Isernia G, Simonte G, Verzini F, Parlani G, et al. Effect of stentgraft model on aneurysm shrinkage in 1,450 endovascular aortic repairs. Eur J Vasc Endovasc Surg 2013;46:192-200.
6. Chaikof EL, Brewster DC, Dalman RL, Makaroun MS, Illig KA, Sicard GA, et al; Society for Vascular Surgery. The care of patients with an abdominal aortic aneurysm: the Society for Vascular Surgery practice guidelines. J Vasc Surg 2009;50(4 Suppl):S2-49.
7. Kasirajan K, Matteson B, Marek JM, Langsfeld M. Technique and results of transfemoral superselective coil embolization of type II lumbar endoleak. J Vasc Surg 2003;38:61-6.
8. Görich J, Rilinger N, Sokiranski R, Krämer S, Schütz A, Sunder-Plassmann L, et al. Embolization of type II endoleaks fed by the inferior mesenteric artery: using the superior mesenteric artery approach. J Endovasc Ther 2000;7:297-301.
9. Nolz R, Teufelsbauer H, Asenbaum U, Beitzke D, Funovics M, Wibmer A, et al. Type II endoleaks after endovascular repair of abdominal aortic aneurysms: fate of the aneurysm sac and neck changes during long-term follow-up. J Endovasc Ther 2012;19:193-9.
10. Jones JE, Atkins MD, Brewster DC, Chung TK, Kwolek CJ, LaMuraglia GM, et al. Persistent type 2 endoleak after endovascular repair of abdominal aortic aneurysm is associated with adverse late outcomes. J Vasc Surg 2007;46:1-8.
11. van Marrewijk CJ, Fransen G, Laheij RJ, Harris PL, Buth J; EUROSTAR Collaborators. Is a type II endoleak after EVAR a harbinger of risk? Causes and outcome of open conversion and aneurysm rupture during follow-up. Eur J Vasc Endovasc Surg 2004;27:128-37.
12. Sampaio SM, Panneton JM, Mozes GI, Andrews JC, Bower TC, Kalra M, et al. Aneurysm sac thrombus load predicts type II endoleaks after endovascular aneurysm repair. Ann Vasc Surg 2005;19:302-9.
13. Faries PL, Briggs VL, Bernheim J, Kent KC, Hollier LH, Marin ML. Increased recognition of type II endoleaks using a modified intraoperative angiographic protocol: implications for intermittent endoleak and aneurysm expansion. Ann Vasc Surg 2003;17:608-14.
14. AbuRahma AF, Mousa AY, Campbell JE, Stone PA, Hass SM, Nanjundappa A, et al. The relationship of preoperative thrombus load and location to the development of type II endoleak and sac regression. J Vasc Surg 2011;53:1534-41.
15. Gallagher KA, Ravin RA, Meltzer AJ, Khan MA, Coleman DM, Graham AR, et al. Midterm outcomes after treatment of type II endoleaks associated with aneurysm sac expansion. J Endovasc Ther 2012;19:182-92.
16. Sheehan MK, Hagino RT, Canby E, Wholey MH, Postoak D, Suri R, et al. Type 2 endoleaks after abdominal aortic aneurysm stent grafting with systematic mesenteric and lumbar coil embolization. Ann Vasc Surg 2006;20:458-63.
17. Gelfand DV, White GH, Wilson SE. Clinical significance of type II endoleak after endovascular repair of abdominal aortic aneurysm. Ann Vasc Surg 2006;20:69-74.
18. Bastos Gonçalves F, van de Luijtgaarden KM, Hoeks SE, Hendriks JM, ten Raa S, Rouwet EV, et al. Adequate seal and no endoleak on the first postoperative computed tomography angiography as criteria for no additional imaging up to 5 years after endovascular aneurysm repair. J Vasc Surg 2013;57:1503-11.
19. Jonker FH, Aruny J, Muhs BE. Management of type II endoleaks: preoperative versus postoperative versus expectant management. Semin Vasc Surg 2009;22:165-71.
20. Nevala T, Biancari F, Manninen H, Aho PS, Matsi P, Mäkinen K, et al. Type II endoleak after endovascular repair of abdominal aortic aneurysm: effectiveness of embolization. J Cardiovasc Intervent Radiol 2010;33:278-84.
21. Patatas K, Ling L, Dunning J, Shrivastava V. Static sac size with a type II endoleak post-endovascular abdominal aortic aneurysm repair: surveillance or embolization? Interact Cardiovasc Thorac Surg 2012;15:462-6.
22. Steinmetz E, Rubin BG, Sanchez LA, Choi ET, Geraghty PJ, Baty J, et al. Type II endoleak after endovascular abdominal aortic aneurysm repair: a conservative approach with selective intervention is safe and cost-effective. J Vasc Surg 2004;39:306-13.

Submitted Aug 12, 2013; accepted Oct 22, 2013.